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In pre-school children, sleep objectively assessed via actigraphy remains stable over 12 months and is related to psychological functioning, but not to cortisol secretion

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ABSTRACT

Study objectives: Studies of the long-term stability of sleep in pre-schoolers are scarce. The aim of the present study was to investigate objectively assessed sleep via actigraphy in pre-schoolers longitudinally, and to predict sleep, psychological functioning and cortisol secretion prospectively as a function of sleep 12 months earlier.

Method: A total of 73 pre-schoolers (mean age: 5.45 years; 53% females) were assessed again after 12 (mean age: 6.4 years). Sleep-actigraphy recordings were performed, saliva cortisol was analysed, and parents and experts rated children's psychological functioning.

Results: Longitudinally, poor sleep at age 5.45 years was associated with poor sleep and internalizing and peer problems but not with externalizing problems and hyperactivity, and cortisol secretion 12 months later. At age 6.4 years and cross-sectionally, poor sleep was concurrently associated with greater psychological difficulties and increased cortisol secretion.

Conclusion: In pre-schoolers, poor sleep objectively assessed at age five was associated with psychological difficulties and poor sleep as assessed via actigraph and one year later. Results indicate that in pre-schoolers sleep remains stable over a 12-mont interval. Pre-schoolers with poor sleep appear to be at risk for developing further psychological difficulties.

1. Introduction

In childhood and adolescence poor sleep, understood as disrupted and non-restoring sleep, is associated with externalizing (aggression and conduct problems) and internalizing problems (social withdrawal; symptoms of anxiety and depression; Aronen et al., 2000; Bernier et al., 2013; Gregory and Sadeh, 2012). Though, in these regards, very few longitudinal studies on the relationship between poor sleep and poor psychological functioning in later life have been conducted.

For sleep difficulties during childhood, Wong et al. (2010) showed that sleep difficulties at kindergarten age predicted alcohol-related problems in young adulthood. Zhang et al. (2010) found that children suffering from chronic sleep difficulties at age nine were at increased risk for poor general health and risky lifestyle practices at age thirteen. Gregory et al. (2009) showed that sleep problems during childhood predicted poor neuropsychological performance during adolescence. Touchette et al. (2012) found poor sleep at age ten to be associated with a higher prevalence of externalizing problems 20 years later.

For sleep difficulties during adolescents, El-Sheikh et al. (2010) showed that in a sample of 141 eleven year olds, sleep problems were associated both cross-sectionally and longitudinally with poorer adjustment outcomes. Similarly, Kaneita et al. (2009) followed up 516 eleven years olds two years later. They observed that

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the onset of poor health status was significantly associated with persisting and with emerging sleep disturbance. Likewise, Roberts et al. (2008) followed-up 3434 11–17 year olds one year later; the key findings were that participants reporting poor sleep at the first time point also complained about poor sleep 12 months later and that persisting poor sleep predicted poor physical and mental health.

To summarize, the existing literature suggests that poor sleep during childhood and adolescence may persist and may be associated with unfavourable physical and psychological functioning. However, long-term studies focussing on pre-schoolers and assessing a key feature of physiological arousal, namely cortisol secretion, do not exist.[2] The aim of the present study was to fill this gap.

The present study expands upon the existing literature in six ways. 1) In contrast to large-scale surveys using self-ratings, parent and teacher rating questionnaires (for exception see El-Sheikh et al., 2010), 2) we assessed a smaller sample of pre-schoolers, 3) using objective, actigraphy recordings, 4) together with emotional, cognitive and behavioural data derived from parents' and experts' ratings, and 5) we also assessed salivary cortisol secretion as a marker for the so-called HPA system (hypothalamic-pituitary-adrenocortical system; cf. Holsboer and Ising, 2010). Last, 6) the study covered a time interval of 12 months.

The present investigation builds on two previous longitudinal studies of pre-schoolers. In the first study (Hatzinger et al., 2013a) we showed that cortisol secretion remained stable over a time interval of 12 months. Importantly, cortisol secretion over time was related to gender – it increased more among girls - and to increased psychological difficulties. In the second study (Hatzinger et al., 2013b) we assessed pre-schoolers' sleep via sleep-EEGs, and we showed that sleep remained stable over time and was related to psychological functioning, but not to cortisol secretion. Importantly, at the age of 5.4 years, participants could be assigned to sub-groups of poor, normal and good sleepers (see below), and this assignment could be replicated at the age of 6.4 years.

The present study builds upon our previous research in that children's sleep was objectively assessed via actigraphs 12 months later. This enabled calculation of the stability of pre-schoolers' sleep as assessed via actigraphs over time. It also allowed us to relate sleep to emotional and behavioural dimensions and to cortisol secretion (again, 12 months later), and to estimate to what extent actigraphs might be applied as.

The following four hypotheses were formulated. Following others (e.g., Roberts et al., 2008; Hatzinger et al., 2013b; Touchette et al., 2012), we expected first that sleep stability at time one would be associated with sleep quality 12 months later, second that sleep stability at time one would be associated with psychological difficulties 12 months later and third that sleep stability at time one would be associated with cortisol secretion. Fourth, following previous research (e.g. Hatzinger et al., 2007, 2008), we anticipated that sleep stability would be associated concurrently with the extent of psychological difficulties and with cortisol secretion level.

[2] A wealth of studies show the close association between poor sleep and an increased cortisol secretion (Steiger et al., 2013; Buckley and Schatzberg, 2005) for instance in infants (Brand et al., 2011, 2014), preschoolers (Hatzinger et al., 2008, 2010, 2012), normal children (Fernandez-Mendoza et al., 2014), children suffering from obstructive apnea syndrome (Patacchioli et al., 2014), healthy adults volunteers (Hori et al., 2011), and in various clinical and non-clinical samples (Steiger, 2002).

2. Methods

2.1. Study population

As described in detail elsewhere (Hatzinger et al., 2007, 2008, 2010), 102 children (mean age 4.91 years; SD = 0.44) starting in kindergarten were recruited from 18 kindergartens in Basel (Switzerland). Children were enrolled in the study after a thorough clinical examination in order to exclude those with any relevant medical and/or neurological disorders. None of the participants had been subjected to sleep deprivation, time shifts or intake of any disturbing substances/medication during the three months prior to the investigation. From the initial 102 children, 82 (49 boys and 33 girls; mean age: 5.91 years) agreed to undergo actigraphic sleep profile analysis (Hatzinger et al., 2008).

Twelve months later, 73 out of these 82 children (89% of the initial sample: mean age: 6.51 years; SD = 0.25; 39 girls (53.4%) and 34 boys (46.6%)) took part in the follow-up study. Again, as described elsewhere (Hatzinger et al., 2013b), all children were briefly medically examined and all children continued to attend the kindergarten regularly. Data were collected during the spring. Finally, parents reported that children were sleeping alone in their own beds.

2.2. Study design

Assessment consisted of three main parts (see Hatzinger et al., 2010, 2013b). Briefly, children's sleep was assessed objectively using an actigraph for seven consecutive nights. On the morning of the last night, four saliva samples were taken to assess the hypothalamic–pituitary–adrenocortical axis activity (HPA AA) via saliva cortisol at baseline conditions. To assess the HPA AA under challenge conditions, six saliva samples were taken while children underwent a non-pharmacological stress test. Experts assessed children's psychological functioning via the Berkley Puppet Interview (Measelle et al., 1998). Parents completed the Strengths and Difficulties Questionnaire (Goodman, 1997).

The purpose and experimental details of the study were fully explained to the children and their parents. The parents were asked to sign an informed consent form before their children entered the study. The experimental protocol was carried out in accordance with the Declaration of Helsinki, and was approved by the local ethics committee.

2.3. Assessment of children's behavioural/emotional difficulties and competences

Parental report of difficulties/competences (Strengths and Difficulties Questionnaire (SDQ), Goodman, 1997).

Children's difficulties/competences were assessed on five scales: emotional problems, conduct problems, hyperactivity, peer problems and pro-social behaviour. Each scale consists of 5 items that are rated on a three-point scale. Internal consistency was moderate to high (Cronbach's $\alpha = .79$).

Children's inner world: experts' ratings based on self-reported difficulties/competences.

The Berkeley Puppet Interview (BPI, Measelle et al., 1998) involves two identical hand puppets making opposing statements on a topic, following which the child can give his/her own view. The interview is videotaped and the recordings scored by independent raters. Each item is rated on a 7-point scale. The following scales were assessed: depression, separation anxiety, over-anxiousness, oppositional/defiant, overt aggression to peers, impulsivity, pro-social behaviour and peer victimization (i.e., being the target of peer aggression or bullying).

2.4. Sleep assessment

As previously reported (Hatzinger et al., 2010), sleep was assessed with two instruments. First, parents were trained to keep a daily log of their child's sleep. They indicated the days of the week and the time when the child went to bed and when she or he woke up. Moreover, parents noted when the actigraph was not worn (e.g., when swimming or bathing), if the child was napping, and if any change in sleep routine occurred. The information derived from the daily sleep log was also used to check possible discrepancies or missing data from the actigraph.

Second, sleep was objectively assessed under at-home conditions for seven consecutive days and nights. According to Sadeh et al. (1991), at-home sleep assessment in children has the advantage that sleep does not seem to be negatively affected. Children wore a digital movement-measuring instrument (actigraph; Somnowatch®; Somnomedics, Randersacker, Germany) on the wrist of the non-dominant hand. This commercially available tool has the dimension of a wristwatch, and it registers every movement above 0.012 g in a bi-axial direction. The data, recorded at 30-s intervals, were digitally integrated and afterwards translated into sleep measures using the software program (based on sleep/wake algorithm as defined by Gorny et al., 1997) provided by Somnowatch®, Somnomedics, Germany. First, parents and children were instructed in how to wear and use the device. They were trained to push the 'marker button' to indicate the beginning and the end of the night, that is to say, in the evening at lights-out, a parent or the child pushed the button to indicate bedtime. In the morning, as soon as the child was awake, the child pushed the button again to indicate end of sleep time. If unsure whether they had pushed the button firmly enough, parents and children were instructed to push the button a second time. To become accustomed to the wristwatch-like actigraph, children started wearing the instrument during an afternoon. This avoided possible first-night effects, though first-night effects with actigraphs have not so far been reported (cf. Sadeh et al., 1991).

Following Sadeh et al. (2000), the following sleep–continuity parameters were computed: sleep onset latency (SOL) as the difference between the first minute of three consecutive minutes of sleep after reported bedtime and the reported bedtime (i.e., the marker indicated by pushing the button in the evening); morning awakening time as the last minute identified as sleep (i.e., the marker indicated by pushing the button in the morning); the number and the times of awakenings after sleep onset (SO); sleep period time (SPT), that is, the sleep time (minutes) from SOL to morning awakening. The following dependent variables were derived from these parameters: total sleep time (TST) being the number of minutes of sleep time excluding all time awake; SOL, SPT, time and number of awakenings after sleep onset (SO), and the sleep efficiency (SE), i.e., the ratio of TST to SPT.

2.5. HPA-system assessment

2.5.1. HPA system activity under baseline conditions

As outlined elsewhere (Hatzinger et al., 2013a), HPA axis activity was assessed by saliva cortisol measures which were sampled during regular week days before Kindergarten lessons. The morning cortisol response with strict reference to the time of awakening has been shown to be a reliable index of basal HPA axis activity and has been described in detail elsewhere (e.g., Pruessner et al., 1997). Four saliva cortisol samples were taken by the parents in the morning at 0, 10, 20, and 30 min after the child awoke. Waking time ranged from 6.50 to 7.30 am. Samples were taken not later than three weeks after the assessment of cortisol levels under stress conditions. Saliva cortisol samples were collected the morning after the sleep-EEG registration.

2.5.2. HPA system activity under stress conditions

Since for preschool children there is currently no non-pharmacological stress test demanding high ego involvement and leading to a reliable and reproducible hormonal response, we used the MacArthur Story Stem Battery (MSSB; as described in detail elsewhere: Von Klitzing et al., 2003; Hatzinger et al., 2007, 2013a, 2013b). Cortisol samples were taken during the MSSB-task as described below. MSSB tests were conducted in the kindergarten, thus depending on a fixed schedule. Subsequently, further tests including the Berkley Puppet Interview (BPI) were conducted.

2.5.3. Saliva cortisol sampling technique and cortisol analysis

Saliva cortisol sampling techniques and cortisol analyses have been described in detail elsewhere (Hatzinger et al., 2013a). Briefly, saliva samples were obtained using the "Salivette" device for quick and hygienic sampling (Sarstedt, Nümbrecht/Germany). This device includes a small cotton swab on which the subject gently chews for 0.5–1 min. Thereafter, the swab is transferred into a small plastic tube, the Salivette container, and stored in the freezer. For morning cortisol measurements, saliva sampling was started immediately after awakening without first rinsing the mouth with water. Participants were asked not to eat breakfast or to brush their teeth before sampling was completed. Saliva samples were returned to the laboratory where they were centrifuged at 4 °C (2000 rpm, 10 min) and stored at −20 °C until assay.

Free salivary cortisol concentrations were analysed using a time-resolved immunoassay with fluorometric detection "Coat-A-Count" Cortisol RIA from DPC (Diagnostics Products Corporation; Biermann GmbH, Bad Nauheim, Germany) as described in detail elsewhere (Dressendörfer et al., 1992). Intra- and interassay variability of this assay was less than 2.00% and 2.04%.

2.6. Statistical analyses

2.6.1. Longitudinal statistical computations

For the first assessment (Hatzinger et al., 2010), based on objectively assessed sleep variables, children were categorised as poor (19.5%; $n = 16$), normal (58.5%; $n = 48$) or good (22%; $n = 18$) sleepers. To this end, in accordance with Sadeh et al. (2000) and with the DSM-IV criteria for insomnia sleep disorders (DSM-IV, APA, 2000), participants were clustered with respect to their baseline values for the following three variables as a starting point for cluster analysis: SOL (sleep onset latency), SPT (sleep period time) and the total time of awakenings after sleep onset. Cluster analyses were performed applying the method of linkage between groups and Euclidean distance as measure (Anderberg, 1973). This factor, Group, served as independent variable in a series of ANOVAs while current sleep variables, psychological assessment variables and cortisol secretion (baseline and challenge conditions) at the second time point 12 months later were introduced as dependent variables. Post-hoc analyses for multiple testing were performed with p-values corrections after Games-Howell, as this procedure does not depend on equal sample sizes.

In addition, at time two after 12 months, sleep variables were also clustered into poor (21.9%; $n = 16$), normal (53.4%, $n = 39$), and good (24.7%, $n = 18$) sleepers, applying the algorithm as described above. To compare distributions and to explore the stability of the group assignments over time, a Chi-square test was performed. Associations between single sleep variables and cortisol values and also between difficulties/competences and sleep variables were computed with Pearson's r correlations.

Table 1
Descriptive and inferential statistics of objective sleep variables at the age of 6.4 years, separately by clusters of poor, normal and good sleepers at the age of 5.4 years.

	Clusters			Statistical analysis	
	Poor [P]	Normal [N]	Good [N]	ANOVA	Group comparisons
N (%)	16 (21.9)	39 (53.4)	18 (24.7)	*F*	
SPT (min)	527.54 (25.47)	582.19 (25.02)	621.64 (14.25)	16.56***	G > N > P
SOL (min)	28.12 (13.95)	15.31 (10.09)	9.46 (12.78)	9.46***	G > N > P
TST (min)	489.42 (16.98)	566.88 (19.45)	612.18 (13.97)	10.27***	G > N > P
WASO-T (time in min)	19.46 (9.20)	10.47 (7.27)	8.32 (1.86)	7.07***	G > P, N > P
WASO-N (number)	6.45 (4.26)	3.05 (2.59)	1.12 (0.26)	7.59**	G > P, N > P
Sleep efficiency (%)	92.77 (2.46)	97.37 (1.98)	98.47 (1.46)	3.49*	G > P, N > P

Notes: SPT = sleep period time; SOL = sleep onset latency; WASO-T = time of awakenings after sleep onset; WASO-N = number of awakenings after sleep onset; ANOVAs: degrees of freedom: always (2, 71); ** = $p < .01$; *** = $p < .001$; single post-hoc group comparisons after Games-Howell with *p*-corrections for multiple testing.

2.6.2. Cortisol samples

The hormone concentration of the morning cortisol samples at +0, +10, +20 and +30 min after awakening was computed as the area-under-the-concentration-time-curve (AUC; using the trapezoidal integration; Forsythe et al., 1969) The AUC total refers to the entire amount of cortisol concentration under the time curve, whereas the AUC basal describes the initial and averaged amount of cortisol secretion over time, as if the HPA-axis had not been stimulated. Accordingly, AUC netto reflects the difference between AUC total and AUC basal.

An alpha of below 0.05 was accepted as nominal level of significance. For multiple testing, the alpha level was adjusted accordingly as indicated above. Statistics was performed with SPSS® 20.0 (IBM Corporation, Armonk NY, USA) for Apple MacIntosh®.

3. Results

3.1. Clusters of poor, normal and good sleepers at age five and sleep variables at age six (longitudinal analysis)

Table 1 gives descriptive and inferential statistics for the sleep variables at age 6.4 years, separately by clusters of poor, normal and good sleepers previously established at age 5.4 years.

There were significant differences on all sleep variables. Post-hoc analyses after Games-Howell showed that compared to good sleepers at time one, poor sleepers had more unfavourable sleep values at time two. Also, normal sleepers at time one had more favourable sleep values than poor sleepers at time two.

Results from the chi-square test showed that poor, normal or good sleepers at age 5.4 years were more likely to be also poor, normal and good sleepers respectively at age 6.4 years ($X^2(N = 73, df = 2) = 57.06$, $p < .001$).

Table 2
Descriptive and inferential statistics of cortisol secretion at baseline (morning cortisol) and under challenge conditions at the age of 6.4 years, separately by clusters of poor, normal and good sleepers at the age of 5.4 years.

	Clusters			Statistical analysis
	Poor [P]	Normal [N]	Good [N]	ANOVA
N (%)	18 (21.9)	39 (53.4)	16 (24.7)	*F*
Baseline				
AUC basal	35.79 (19.42)	26.01 (15.21)	36.12 (25.46)	1.02
AUC total	53.46 (8.31)	52.98 (12.45)	49.21 (10.98)	1.11
AUC netto	17.67 (14.35)	26.97 (13.22)	13.09 (19.25)	0.90
Challenge				
AUC basal	56.53 (23.73)	64.12 (13.62)	75.65 (36.02)	1.23
AUC total	98.12 (16.42)	73.16 (16.99)	86.02 (42.06)	0.03
AUC netto	41.59 (31.01)	9.04 (13.10)	10.37 (35.32)	0.61

Notes: AUC = area under the curve. ANOVAs: degrees of freedom: always (2, 71).

3.2. Sleep at age 5 and psychological functioning 12 months later (longitudinal analysis)

Using the SDQ, parents of poor sleepers at age 5.4 rated them as a) having more internalizing problems at age 6.4 ($M = 0.83$; SD = 0.21; $F(2, 71) = 5.13$, $p < .01$; compared to good sleepers ($M = 0.18$; SD = 0.12; [normal sleepers: $M = 0.30$; SD = 0.18]), and b) having more negative peer relationships ($M = 0.95$; SD = 0.25; $F(2, 71) = 4.89$, $p < .01$; compared to good sleepers ($M = 0.12$; SD = 0.09; [normal sleepers: $M = 0.35$; SD = 0.21]), while c) their ratings summed to higher total scores (good sleepers: $M = 0.20$; SD = 0.17; normal sleepers: $M = 0.36$; SD = 0.20; poor sleepers: $M = 0.68$; SD = 0.29; $F(2, 71) = 6.41$, $p < .01$). No significant group differences were found for externalizing problems, hyperactivity, or prosocial behaviour.

With respect to children's inner world as assessed by the Berkley Puppet Interview (BPI), compared to good sleepers ($M = 5.85$, SD = 0.42; [normal sleepers: $M = 4.23$, SD = 0.46]), poor sleepers $M = 3.98$; SD = 1.45; $F(2, 71) = 5.85$, $p < .05$) at age 5.4 years had significantly higher social inhibition and less prosocial behaviour at age 6.4 years (good sleepers; $M = 5.75$, SD = 0.65; normal sleepers: $M = 5.45$, SD = 1.02; poor sleepers $M = 4.03$; SD = 0.85; $F(2, 71) = 6.00$, $p < .05$). For all other dimensions (depression, anxiety, oppositional/defiant, overt aggression to peers, impulsivity, victimization), no significant group differences were observed.

3.3. Sleep clusters at the age of 5 years and cortisol secretion 12 months later (longitudinal analysis)

Table 2 gives descriptive and inferential statistics for current cortisol secretion (baseline and challenge conditions), separately by categories of poor, normal and good sleepers at age 5.4 years.

Cortisol secretion at baseline and under challenge conditions at 6.4 years did not differ significantly as a function of sleep category 12 months earlier.

3.4. Associations between sleep characteristics, psychological functioning and cortisol secretion at 6.4 years (cross-sectional analysis)

Prolonged sleep onset latency ($r = 0.25$*[3]), more frequent awakenings after sleep onset ($r = 0.33$*), and longer time of awakenings after sleep onset ($r = 0.29$*) were associated with increased cortisol secretion in the morning (baseline; AUC netto).

For cortisol secretion under challenge conditions, there were no statistically significant associations.

[3] * = $p < .05$; ** = $p < .01$.

For the association between sleep dimensions and psychological dimensions, the following pattern of results was observed. Both a longer sleep onset time and a greater number of awakenings after sleep onset were associated with greater hyperactivity (SDQ; $r = 0.28^*$; $r = 0.31^*$), negative peer relationships (SDQ; $r = 0.34^*$; $r = 0.32^*$), and overt aggression to peers (BPI; $r = 0.34^*$; $r = 0.29^*$). No other associations were significant.

Overall, at 6.4 years poor sleep was associated with higher cortisol secretion (baseline) and more unfavourable psychological functioning.

4. Discussion

The key findings of the present study are that in a sample of pre-schoolers, objectively assessed sleep via actigraphy remained stable over a 12 month period, and that psychological functioning (internalizing problems, peer relationship, but not externalizing problems and hyperactivity) at age 6.4 years varied as a function of sleep patterns 12 months earlier, whereas cortisol secretion at 6.4 years was not associated with sleep at 5.4 years. In addition, poor sleep, unfavourable psychological functioning and higher cortisol secretion were related to one another at six years, that is, cross-sectionally. In our opinion, the present findings add to the current literature in that the pattern of longitudinal and cross-sectional associations between sleep, psychological functioning and cortisol secretion were based on data gathered from actigraphy, an easily applicable and cost-effective device to assess sleep objectively. Moreover, the pattern of results is similar to the results gathered via sleep-EEGs (Hatzinger et al., 2013b), suggesting again that actigraphs seem to be a valuable and cost-effective device to gather sleep data.

Four hypotheses were formulated and each of these is considered in turn.

Our first hypothesis was that poor sleep at five years would be associated with poor sleep at six years, and this hypothesis was supported: children categorized respectively as poor, normal and good sleepers at five years were more likely to be assigned to the corresponding categories at six years. In this respect, the present pattern of results accords with previous research (e.g. Roberts et al., 2008; Hatzinger et al., 2013b). However, the present study confirms (Hatzinger et al., 2013b) and expands upon previous research in that this pattern of association was found among five to six years olds using objective measures of sleep. Moreover, we showed that among pre-schoolers, sleep remained fairly stable over a period of 12 months.

Second, we hypothesized that sleep patterns at five years would be associated with psychological difficulties and our data broadly confirmed this. In this respect the present findings are consistent with other research using questionnaire-based assessments and sampling different ages. Zhang et al. (2010) showed that 9 years olds with severe sleep difficulties also had an increased risk for poor general health at age 13. Data from the present study further validates previous findings (2013b) in that sleep was objectively assessed and children's psychological functioning was assessed both by parents' and experts' ratings.

Our third hypothesis was that sleep patterns at age five would be associated with cortisol secretion at age six and, as in a previous study (Hatzinger et al., 2013b), this hypothesis was not supported. From the data available to us we cannot say with any certainty why the expected association was not observed, though it is possible that either 1) the sample was too small for the statistical power needed to detect an association, 2) unknown and unassessed variables confounded possible associations; especially the HPA AA is dependent from several factors and shows therefore much lighter state variability than other neuroendocrine systems, or 4) there is effectively no neurophysiological or neuroendocrine relation between sleep at five years and cortisol secretion at six years. In this respect, we note that the development and adaptation of the HPA axis is still subject to changes in physiological development at these ages (Essex et al., 2011) and that HPA axis activity at six years might be modified irrespective of sleep quality 12 months earlier. Moreover, as the present data showed (see fourth hypothesis) that changes in HPA axis activity were significantly associated with current sleep and psychological functioning. In our opinion, these findings are in accord with results from adult studies indicating that HPA AA is a state rather than a trait marker (cf. Ising et al., 2005).

Our fourth hypothesis was that sleep quality would be concurrently associated with psychological difficulties and cortisol secretion levels. Data did generally support this hypothesis. In this respect, we were able to replicate previous findings (e.g., Hatzinger et al., 2008, 2012, 2013b; Steiger et al., 2013), but based on the greater validity of objective assessment of sleep quality.

The question arises as to why sleep, psychological functioning and cortisol secretion should be associated. The answer is likely to be complex and beyond a mere cause-effect-relation.

First, numerous studies show that sleep restriction adversely affects cognitive, emotional and behavioral performance. For example, Gruber et al. (2012), assigned 34 children aged 7–11 years either to a cumulative sleep-restricted (−54 min/night respect to baseline) or to a cumulative sleep-extended (+28 min/night respect to baseline) condition. They found that, relative to sleep restriction, a modest extension in sleep duration led to significant improvements in alertness and emotional regulation. From this study we note that changes in sleep have a causal impact on emotion, cognition and behaviour. The present study indicates that poor sleep may unfavourably influence both children's psychological functioning and their sleep quality 12 months later.

Taking sleep as the starting point, Hori et al. (2011) found increased cortisol reactivity during a pharmacological challenge of the HPA axis in healthy adult volunteers as a function of sleep quality and sleep quantity. The authors concluded that poor sleep was associated with exaggerated cortisol reactivity. The present study indicates that poor sleep may unfavourably influence children's concurrent HPA axis activity, but not over a time interval of 12 months.

Taking psychological processes as a starting point, Born et al. (1999) showed that timing the end of sleep by mere volition was causally linked to increased cortisol secretion. From this study we note that psychological processes can alter sleep and, most importantly, neuroendocrine processes such as cortisol secretion. Accordingly, it is possible that in the present study unfavourable psychological functioning might have caused or maintained concurrent poor sleep and increased cortisol secretion.

Last, taking cortisol secretion as a starting point, a large body of research on depressive disorders indicates that a deteriorated HPA axis can precede both the onset and the recurrence of a depressive disorder (Hatzinger et al., 2002; Holsboer and Ising, 2010). Thus it is possible that our results reflect unfavourable negative influence of increased HPA axis activity on children's current sleep and psychological functioning.

To summarize, the above studies indicate that sleep, psychological functioning and cortisol secretion are highly intertwined. From the present data we see that these associations also hold for five- to six years olds assessed both cross-sectionally and longitudinally.

Despite the intriguing results, several limitations warrant caution in generalizing from these findings. First, only parents and children willing and able to complete the entire study protocol took part in the study, and systematic sample-related biases cannot be excluded. Second, the sample of 73 children who completed the longitudinal study might be rather small. This is potentially a

problem with respect to the psychological/psychosocial variables and greater statistical power would have provided a more robust test of hypotheses. In these respects, we also note a number of non-significant findings. Data, unfortunately, do not allow a deeper introspection to explain the number of non-significant findings (namely the lack of associations between sleep at the age of 5 years and externalizing problems and hyperactivity), though we suspect that either there was really no association or that the statistical power was too low to detect more meaningful association. Consequently, generalization with respect to psychological/psychosocial values should be made with particular caution. Third, the present pattern of result might have emerged due to further latent, though not assessed factors. Last, we are fully aware that the sleep pattern at time one would have been modified by psychological and neuroendocrine processes (Hatzinger et al., 2008), though here we decided to focus on the association of sleep patterns with sleep, cortisol secretion and psychological functioning 12 months later. As for the long-term development of cortisol secretion, we showed that it remained stable over 12 months and was related to psychological functioning and gender (Hatzinger et al., 2013a).

5. Conclusions

Among a sample of pre-schoolers, sleep assessed via actigraphs remained stable over 12 months. Sleep at age of five was associated with sleep and psychological functioning, but not cortisol secretion, at age six. The pattern of results underscores the tight relationships between sleep, HPA axis activity and psychological functioning already observable among pre-schoolers. Moreover, actigraphic sleep assessments provide useful sleep continuity data comparable to sleep continuity data carried out from sleep-EEGs.

Conflicts of interest

All authors declare no conflicts of interest.

Contributors

MH, SB, SP, AvW, KvK, SS, and EHT designed the study and wrote the protocol, and all of them managed the literature searches and analyses. SB, SP, SS and AvW were highly engaged with data collection and data entry. SB undertook the statistical analyses, and SP the statistical check. SB wrote the draft of the manuscript and coordinated the integration of the different comments and corrections of the authors. MH and EHT are SB's senior researchers. KvK is the responsible senior researcher of SP, SS and AvW. All authors contributed to and have approved the final manuscript.

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